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Chapter 25

Polysomnography

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Abstract

Polysomnography refers to a systematic process used to collect physiologic parameters during sleep. A polysomnogram (PSG) is a procedure that utilizes electroencephalogram, electro-oculogram, electromyogram, electrocardiogram, and pulse oximetry, as well as airflow and respiratory effort, to evaluate for underlying causes of sleep disturbances. PSG is considered to be the gold standard for diagnosing sleep-related breathing disorders, which include obstructive sleep apnea (OSA), central sleep apnea, and sleep-related hypoventilation/hypoxia. PSG can also be utilized to evaluate for other sleep disorders, including nocturnal seizures, narcolepsy, periodic limb movement disorder, and rapid eye movement sleep behavior disorder. With recent technological developments, home sleep apnea testing can be done to confirm a diagnosis in patients with a high risk for moderate to severe OSA in the absence of comorbid medical conditions or other suspected sleep disorders.

HISTORY

Polysomnography

The roots of the modern-day polysomnogram (PSG) are credited to the work of Caton (1875), who discovered brain wave activity in animals in 1875. This very early finding led to the description of differences between wakefulness and sleep by Berger (1929), and ultimately contributed to the first continuous overnight EEG recording during sleep (Loomis et al., 1937). Further work by Aserinsky, Kleitman, Dement, and Jouvet in the 1950s established the utility of the combined use of electroencephalogram (EEG), electro-oculogram (EOG), and electromyogram (EMG) to determine various behavioral states in wakefulness and sleep, including rapid eye movement (REM) sleep (Aserinsky and Kleitman, 1953; Dement and Kleitman, 1957; Jouvet et al., 1959). Dement and Kleitman proposed formal nomenclature for various stages of sleep in 1957. Gastaut et al. (1965) published data on respiratory disturbances during sleep. A standardized manual for terminology, techniques, sleep staging, and respiratory event scoring was published in 1968, known as the Rechtschaffen and Kales (R and K) manual (Kales and Rechtschaffen, 1968). The R and K manual remained as the standard PSG staging and scoring system until 2007, when the American Academy of Sleep Medicine (AASM) published its own manual, which is now used as the required standardized system for all AASM-accredited sleep centers and sleep labs (Iber et al., 2007). The AASM manual is revised every few years, with the latest edition being version 2.4 (Berry et al., 2017). While PSG is utilized for the evaluation of a number of sleep disorders, it mainly serves as the gold standard for the diagnosis of obstructive sleep apnea (OSA).

Obstructive sleep apnea

OSA is by far the most common sleep disorder for which diagnostic testing is done in sleep centers.

A number of excellent publications on the history of sleep medicine are available (Lavie, 1984; Dement and Vaughan, 1999; Pelayo et al., 2010; Thorpy, 2011). Broadbent (1877) provided a detailed description of a patient with Cheyne–Stokes breathing and OSA. He described the observation as alarming and commented that the condition was most probably underrecognized.

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Caton (1889) described the mechanism for sleepiness as due to poison that selectively acted on the pharyngeal muscles. Mitchell (1890) described sleep disordered breathing (OSA and central sleep apnea) as specific disorders, distinguishing them from Cheyne–Stokes respiration. Bramwell (1909) first described a patient as a "Pickwick," the obese and somnolent patron in the Pickwickian Club, from Charles Dickens' book *The Posthumous Papers of the Pickwickian Club* (Dickens, 1837). The "Pickwickian Syndrome" was popularized by Osler (1918), but he may have inadvertently delayed recognition of OSA as a distinct disorder from Pickwickian syndrome.

It was not until 1966 that OSA was characterized as a distinct disorder from Pickwickian syndrome. The use of polysomnography provided the first objective assessment of OSA in Pickwickian patients (Jung and Kuhlo, 1965; Gastaut et al., 1966). In these landmark papers, the suggestion that an intrinsic sleep disturbance was the likely cause of daytime sleepiness, and not CO_2 narcosis, was of paramount importance. The finding that tracheotomy was an effective treatment for OSA (Kuhlo et al., 1969) supported the view that upper airway obstruction was the cause of OSA and that respiratory-related arousals were the cause of daytime sleepiness in Pickwickian syndrome patients.

Diagnostic testing involving the continuous running of paper recordings throughout the sleep period was recognized by Christian Guilleminault, who used this method to fully characterize OSA as it evolved across sleep periods, REM, and non-REM sleep (Dement and Vaughan, 1999).

The effectiveness of treatment could also be assessed using PSG. Alternatives to tracheotomy, including upper airway surgery (Fujita et al., 1981; Riley et al., 1986) and continuous positive airway pressure (CPAP) (Sullivan et al., 1981), were developed. CPAP and other iterations of the original technology remain the first-line treatment for OSA patients today.

PROCEDURE

Overview

A routine PSG requires a comprehensive monitoring system to record sleep stages, limb movements, airflow, respiratory effort, heart rate and rhythm, oxygen saturation, and body position. This type of study, also known as a Type I (Level I) sleep study, is done in a sleep lab with a trained sleep technician present throughout the duration of the study. PSGs are primarily used to diagnose sleep-related breathing disorders (SRBDs), including OSA, central sleep apnea, and sleep-related hypoventilation/hypoxia. Positive airway pressure (PAP) titration sleep studies are modified PSGs that evaluate the effectiveness of PAP therapy in treating SRBDs. Additionally, PSGs may be used to diagnose sleep-related seizures, periodic limb movement (PLM) disorder, parasomnias, and central hypersomnias (Kushida et al., 2005).

Sleep staging

Sleep staging is determined using information from the EEG, EOG, and EMG electrodes. Electrical activity from frontal, central, and occipital brain regions, eye movements, and chin EMG are all used to determine the five sleep–wake stages: wake (W), stage 1 (N1), stage 2 (N2), stage 3 (N3), and REM sleep. Vertex waves, K-complexes, sleep spindles, delta waves, and sawtooth waves are EEG characteristics seen in different stages of sleep. Eye movement frequency and contour, as well as changes in chin EMG tone, assist with identifying sleep stages (Williams et al., 1974; Silber et al., 2007; Silber, 2009).

For EEG, electrodes are placed in accordance with the International 10–20 System (Jasper, 1958) at F4, C4, O2, and M1 to give the EEG derivations F4-M1, C4-M1, and O2-M1 (Berry et al., 2017). Back-up electrodes are placed on the corresponding left-side locations to give the EEG derivations F3-M2, C4-M2, and O1-M2, in case electrodes malfunction during the study (Fig. 25.1).

Electrodes are placed at E1 and E2 (1 cm below and 1 cm lateral to the left and right outer canthus, respectively) to give rise to the EOG derivations E1-M2 and

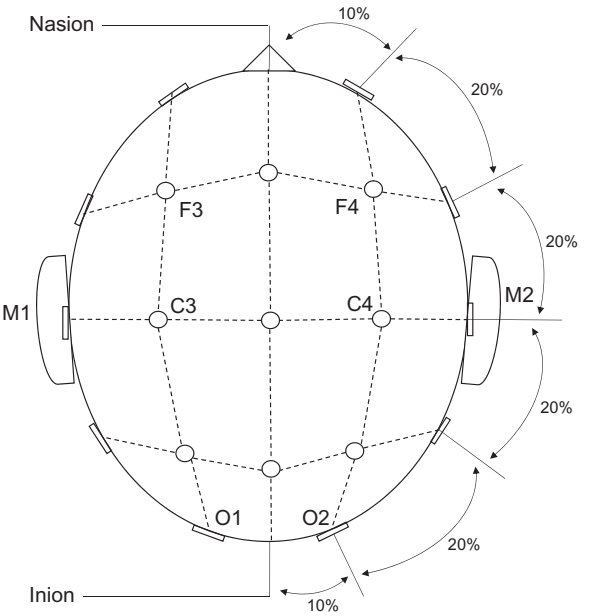

Fig. 25.1. Illustration of electrode placement for recommended derivations for EEG during PSG. Image may not be to scale.

E2-M2. Chin EMG is measured to differentiate REM sleep from wake or other stages of sleep. Low-chin EMG is typically seen with REM sleep muscle atonia. One electrode is placed midline 1 cm above the inferior edge of the mandible with two electrodes placed 2 cm below the edge of the mandible and 2 cm to the left and right of the midline to create submental (chin) EMG derivations. Arousals from sleep stages N1, N2, N3, or REM occur when there is an abrupt shift of the EEG frequency in the alpha or theta range, or frequencies greater than 16 Hz, lasting for at least 3 s. During REM sleep, at least a 1-s increase in chin EMG must also accompany the EEG frequency shift. Arousals are scored throughout sleep to determine the arousal index (level of sleep disruption) and may be used to score some respiratory events (Berry et al., 2017).

Airflow and respiratory effort

Two qualitative airflow sensors are used during PSG. The oronasal thermal sensor (thermistor) detects temperature changes of inspired vs expired breath to monitor airflow and is best used to identify apneas (≥90% reduction in airflow). The nasal pressure transducer detects pressure changes during inspiration and expiration to monitor airflow and is used to identify hypopneas (≥30% reduction in airflow) (Heitman et al., 2002; Redline et al., 2007).

Additionally, two respiratory inductance plethysmography (RIP) sensors or belts are used to determine qualitative respiratory effort in the thorax and abdomen. RIP belts utilize embedded piezocrystals that emit changes in voltage corresponding to movement of the thorax and abdomen during breathing. Collectively, these sensors are used to determine the different types of respiratory events, including obstructive apneas, obstructive hypopneas, and central apneas. Though not routinely included in PSGs, esophageal manometry can be utilized to quantitatively detect increased respiratory effort from upper airway resistance to score respiratory effort-related arousals (RERAs). RERAs are airflow limitation events associated with arousals that do not meet the scoring criteria for hypopneas or apneas.

Oxygen saturation

Oxygen saturation is obtained through pulse oximetry and is used to assist with scoring hypopneas when there is at least a 3% or 4% oxygen desaturation (depending on whether the recommended AASM or acceptable scoring criteria is used) associated with the respiratory event (Berry et al., 2017). Additionally, pulse oximetry data are used to determine whether supplemental nocturnal oxygen is necessary.

Electrocardiogram

A single modified electrocardiograph Lead II with torso electrode placement is used during a PSG to monitor heart rate and rhythm. With a single electrocardiogram (ECG) lead, complex arrhythmias cannot always be defined; however, wide or narrow complex tachycardia, atrial fibrillation, significant heart block, and sustained tachycardia or bradycardia are generally easily recognizable.

Limb EMG

Electrodes are placed on the left and the right anterior tibialis muscles to detect leg movement. Limb movements are scored based on a minimum amplitude increase of 8 μV above the resting EMG voltage with a duration of 0.5–10 s. A PLM series requires at least four consecutive limb movements, with each limb movement in the series between 5 and 90 s apart from the adjacent limb movements.

Body position

Body position is documented in the lab by the sleep technician using video confirmation or by using a position monitor attached to the patient. Recording body position is important in patients with OSA, as respiratory events tend to be more prominent during supine sleep (McEvoy et al., 1986; Oksenberg et al., 2000).

OPTIONAL MEASUREMENTS

Carbon dioxide

While not required, many in-lab PSGs include evaluation of carbon dioxide levels to assess for hypoventilation during sleep. End tidal CO_2 ($EtCO_2$) is measured using a nasal cannula that detects the end of breath CO_2 level during expiration. Alternatively, transcutaneous CO_2 ($TcpCO_2$) can be determined by using electrodes on the skin that measure CO_2 as a product of respiration. $TcpCO_2$ is frequently used in infants and children to avoid artifact and loss of signal that occurs more commonly with $EtCO_2$. Additionally, $TcpCO_2$ may be used during PAP titration studies, as $EtCO_2$ recording is not possible due to placement of the PAP mask. Patients with morbid obesity (BMI > 40 kg/cm^2), neuromuscular disorders, chest wall abnormalities, or pulmonary disorders are more likely to have sleep-related hypoventilation (Sateia, 2014; Boing and Randerath, 2015).

Full EEG montage

A full set of EEG electrodes may be added to the required PSG electrodes to evaluate for possible sleep-related

seizures. An early study using video PSG with full EEG by Aldrich showed that a definitive diagnosis was obtained in 35% of the subjects (Aldrich and Jahnke, 1991). Based on the AASM Standards of Practice, full EEG with PSG and video monitoring is indicated in patients with paroxysmal arousals or sleep disruptions thought to be related to sleep-related seizures when routine clinical evaluation and standard EEGs are negative or inconclusive (Kushida et al., 2005; Foldvary-Schaefer and Malow, 2011). PSG with full EEG is not necessary in patients with paroxysmal arousals typical of non-REM parasomnias, such as sleep walking, sleep terrors, and confusional arousals; or REM parasomnias, such as REM sleep behavior disorder.

Arm EMG

Additional EMG leads placed over the left and right extensor digitorum muscles in the forearms may be used to identify REM without atonia in patients suspected of having REM sleep behavior disorder or other parasomnias with limb movements.

TYPES OF PSGs

Baseline PSG

A typical baseline PSG, also called a Type I (Level I) sleep study, includes the required measurements for EEG, EOG, chin and limb EMG, airflow signals, respiratory effort signals, oxygen saturation, ECG, and body position, to assess for SRBD and other signs of sleep disruptions, such as arousals or PLMs. Optional measurements include $EtCO_2$ or $TcpCO_2$, full EEG, or additional arm EMG leads.

PAP titration study

A PAP titration study is performed to evaluate effectiveness of various PAP therapies and pressure settings to control SRBD. Continuous PAP (CPAP), bilevel PAP, and adaptive servo-ventilation (ASV) can all be tested during a PAP titration study. CPAP is generally the first-line PAP modality for OSA and central sleep apnea. Bilevel PAP is used when there is intolerance to higher pressures of CPAP, persistence of obstructive or central respiratory events, emergence of central events with CPAP, or evidence of hypoventilation. A back-up breathing rate may be added to bilevel PAP or the patient may be transitioned to ASV, if central respiratory events do not resolve with CPAP or bilevel PAP without back-up rate. Required and optional measurements are very similar to the baseline PSG. The main exception is that a PAP lead is used to measure airflow instead of the nasal pressure transducer and thermistor, and $EtCO_2$ cannot be used with PAP therapy because of the location of the sensor and PAP mask.

Split night study

Split night studies combine an initial baseline PSG and a PAP titration study, with combined set-up of leads. The "two studies in one" option allows for diagnosis and treatment evaluation of SRBD in the same night. The AASM recommends this type of study when a diagnosis of severe OSA with an apnea-hypopnea index (AHI) > 40 is obtained within the first couple of hours of the study. Additionally, the study may be "split" before an AHI > 40 is reached if significant O_2 desaturations (<85%) and an AHI > 5 is reached.

METHOD FOR REVIEWING AND INTERPRETING PSGs

Interpretation of the sleep study includes review of the EEG, EOG, and EMG to stage each 30-s epoch of the entire recording. Arousals are also designated when present. The entire study is reviewed again in 2-min epochs to score respiratory events and limb movements. Body position is reviewed using video confirmation or body sensor devices. ECG is reviewed and any abnormalities are described. The sleep hypnogram is a diagram that is used to show an overview of the entire study (Fig. 25.2). A normal sleep study length is 6–8 h, and all sleep stages should be achieved. Sleep study reports should include

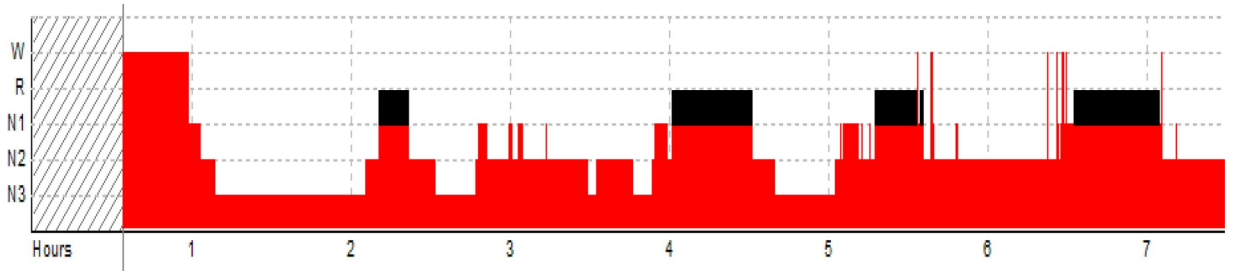

Fig. 25.2. Hypnogram showing normal sleep architecture during a night of sleep with all of the typical sleep stages. W, wake; R, REM sleep; N1, non-REM stage 1 sleep; N2, non-REM stage 2 sleep; N3, non-REM stage 3 sleep.

sleep latency, wake after sleep onset, sleep efficiency (total sleep time divided by total recording time), percentages of all stages of sleep, and arousals from sleep. Additionally, respiratory events and limb movements are reported.

SELECTED PSG ABNORMALITIES

Sleep architecture abnormalities

In-lab PSGs can identify abnormalities in sleep architecture. Sleep latency, calculated as time from start of study to sleep onset, is normally several minutes to 30 min in length. A very short sleep latency during a sleep study may suggest sleep deprivation. A prolonged sleep latency might suggest first-night effect (difficulty sleeping in a new environment) or insomnia (Ong et al., 2017). Additionally, a very high sleep efficiency, as calculated by total sleep time over total recording time, may be seen in patients with sleep deprivation or in patients with idiopathic hypersomnia, a type of central hypersomnia. REM sleep onset typically occurs 90–110 min after sleep onset. An early onset REM period may be seen in patients with sleep deprivation, narcolepsy, or untreated depression. The REM sleep percentage can be increased in patients with untreated depression (Carskadon and Dement, 2017; Minkel et al., 2017).

Respiratory events

Respiratory events during sleep fall may be obstructive or central. Obstructive respiratory events are due to a collapse of the airway, usually in the oropharynx, restricting airflow. Central respiratory events are due to absence of inspiratory effort. Additionally, respiratory events are defined as apneas or hypopneas. Respiratory events must be at least 10 s in length to be scored. To be scored, an apnea must demonstrate at least a 90% decrease in airflow amplitude compared with pre-event baseline in the oronasal thermal sensor (thermistor) during a baseline PSG, or in the device flow signal in a PAP titration study (Berry et al., 2017). Obstructive apneas have continued or increased inspiratory effort for the duration of the apnea, as evidenced in the inspiratory effort chest and abdomen leads (Fig. 25.3).

Central apneas are associated with absence of inspiratory effort (Fig. 25.4). Additionally, mixed apneas can be scored if there is absent inspiratory effort in the initial portion of the apnea, with resumption of inspiratory effort in the latter portion of the apnea. Hypopneas require at least a 30% reduction in airflow amplitude in the nasal pressure transducer or PAP device flow sensor with an associated 3% oxygen desaturation or an EEG arousal (Fig. 25.5). Acceptable scoring criteria for hypopneas, as utilized by some insurance companies, require a 4% oxygen desaturation, without criteria

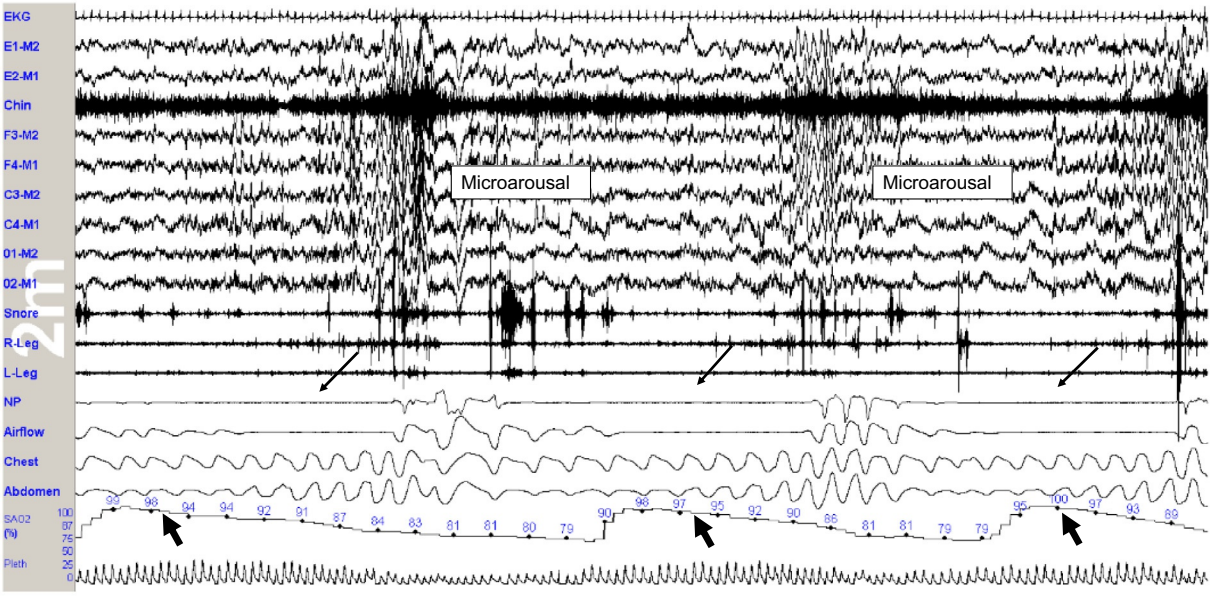

Fig. 25.3. Digital tracing of a 2-min epoch of stage 2 non-REM sleep showing several obstructive apneas (*thin arrows*) with no flow in NP or airflow leads. Effort is present in chest and abdomen leads. There are also associated oxygen desaturations (*wide arrows*) and EEG arousals (microarousal). E1-M2, left EOG; E2-M-1, right EOG; F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1, EEG derivations; chin, chin EMG; EKG, electrocardiogram; snore, snore microphone; R-Leg, right tibialis anterior EMG; L-Leg, left tibialis anterior EMG; NP, nasal pressure transducer; airflow, oronasal thermistor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage; pleth, plethysmography.

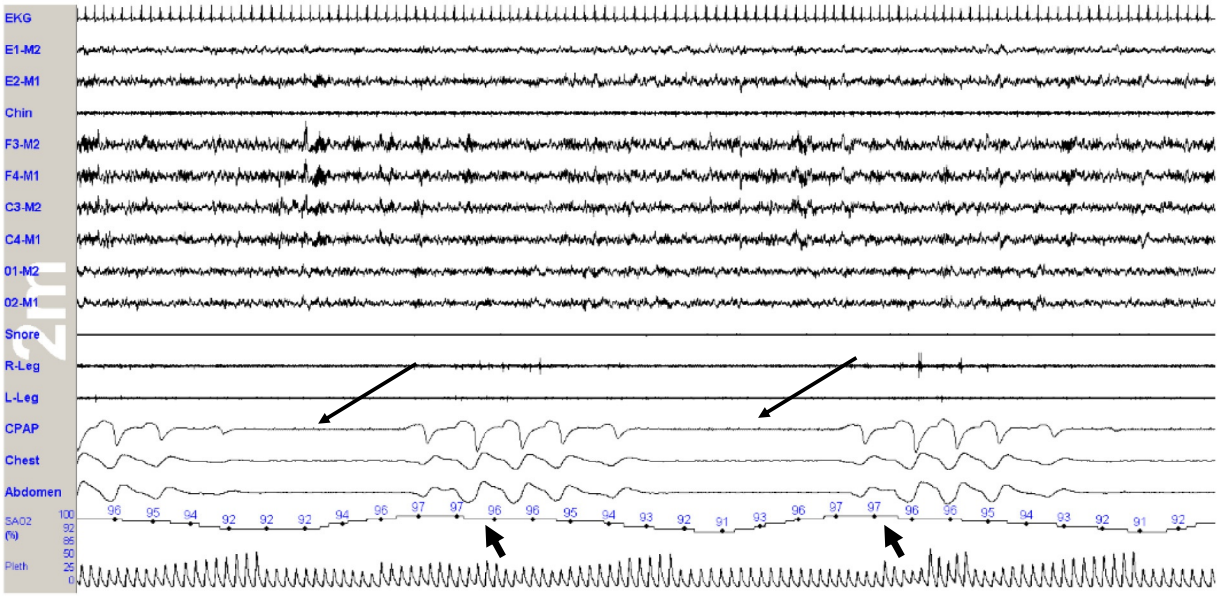

Fig. 25.4. Digital tracing of a 2-min epoch of stage 2 non-REM sleep showing several central apneas (*thin arrows*) during a PAP titration study. There is no flow in the CPAP signal. Effort is not present in chest and abdomen leads. There are also associated oxygen desaturations (*wide arrows*). E1-M2, left EOG; E2-M-1, right EOG; F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1, EEG derivations; chin, chin EMG; EKG, electrocardiogram; snore, snore microphone; R-Leg, right tibialis anterior EMG; L-Leg, left tibialis anterior EMG; NP, CPAP, positive airway pressure airflow sensor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage; pleth, plethysmography.

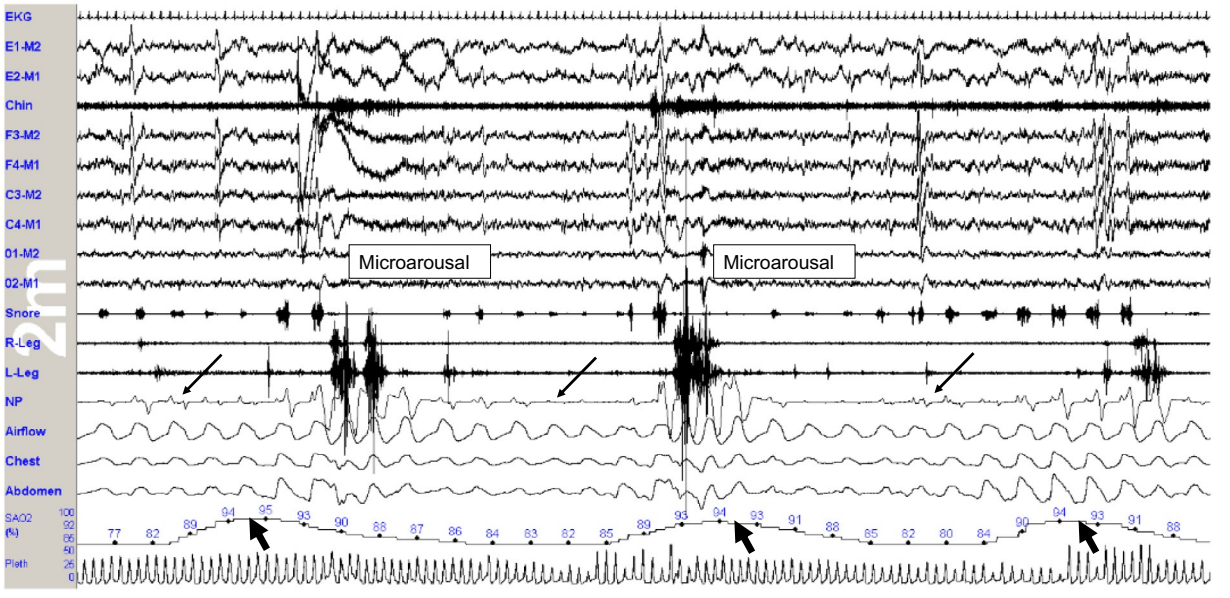

Fig. 25.5. Digital tracing of a 2-min epoch of stage 2 non-REM sleep showing several obstructive hypopneas (*thin arrows*) with reduced flow in NP lead and mildly decreased signal in airflow lead. Effort is present in chest and abdomen leads. There are also associated oxygen desaturations (*wide arrows*) and EEG arousals (microarousal). Limb movements are present after each respiratory event. Snore amplitude decreases during each hypopnea and increases with return of normal breathing. E1-M2, left EOG; E2-M-1, right EOG; F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1, EEG derivations; chin, chin EMG; EKG, electrocardiogram; snore, snore microphone; R-Leg, right tibialis anterior EMG; L-Leg, left tibialis anterior EMG; NP, nasal pressure transducer; airflow, oronasal thermistor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage; pleth, plethysmography.

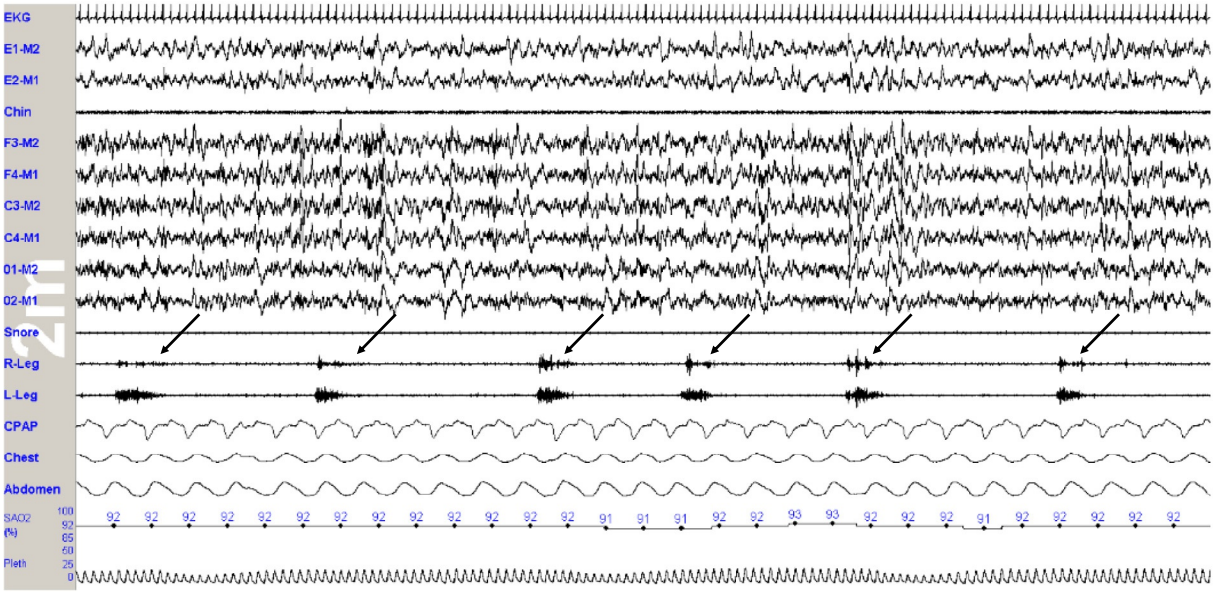

Fig. 25.6. Digital tracing of a 2-min epoch of stage 2 non-REM sleep showing periodic limb movements in the L-leg and R-leg sensors (*arrow*). E1-M2, left EOG; E2-M-1, right EOG; F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1, EEG derivations; chin, chin EMG; EKG, electrocardiogram; snore, snore microphone; R-Leg, right tibialis anterior EMG; L-Leg, left tibialis anterior EMG; NP, nasal pressure transducer; CPAP, positive airway pressure airflow sensor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage; pleth, plethysmography.

related to arousals. Obstructive hypopneas must have associated snoring, increased inspiratory effort, or thoracoabdominal paradoxical breathing pattern during the event. Central hypopneas must not have associated snoring, increased inspiratory effort, or thoracoabdominal paradoxical breathing pattern during the event.

The number of respiratory events divided by the hours of sleep during the sleep study are calculated to determine the AHI. An AHI > 5 is diagnostic of sleep apnea.

Excessive limb movements

Excessive limb movements, as measured by a periodic limb movement index (PLMI) greater than 15, can be seen in a number of disorders, including neuropathy, chronic pain, renal disease, restless legs syndrome, OSA, and in normal aging (Fig. 25.6). Additionally, PLMs may be associated with arousals from sleep, and an elevated periodic limb movement arousal index (PLMArI) indicates that the limb movements are contributing to disruptive sleep. During REM sleep, which is characterized by muscle atonia, transient limb movements may occur during phasic activity; however, excessive limb movements may be seen in patients with REM behavior disorder.

REM without atonia

REM sleep is characterized by muscle atonia, though there may be transient muscle activity in the limbs associated with REMs or in the chin or limb EMG leads with arousals from sleep. REM without atonia is an abnormal finding that is defined as excessive muscle activity in the chin or limb EMG as evidenced by an 8-μV increase in EMG amplitude from baseline, occurring in at least 5 mini-epochs of 3 s each (Fig. 25.7). REM without atonia can be seen with certain medications, including antidepressants (Silber et al., 2017). It also may be seen in primary sleep disorders such as OSA, narcolepsy, and REM behavior disorder. Patients may describe dream enactment behaviors including hitting, punching, kicking, or yelling.

HOME SLEEP APNEA TESTING

Overview

Home sleep apnea testing (HSAT) is an alternative to PSG in the diagnosis of OSA. When used in accordance with the most recent clinical guidelines, HSAT can be part of the assessment and treatment of OSA (Collop et al., 2007; Kapur et al., 2017).

The HSAT is used only for the assessment of OSA in uncomplicated patients who are at moderate to high risk of OSA and who do not have comorbid medical conditions or other suspected sleep disorders (Table 25.1). HSAT is also not recommended in patients over age 65, as it has not been extensively studied in that population, and older patients may have difficulty applying the HSAT sensors properly. Additionally, patients with a body mass index of $>40\,kg/cm^2$ are at increased risk

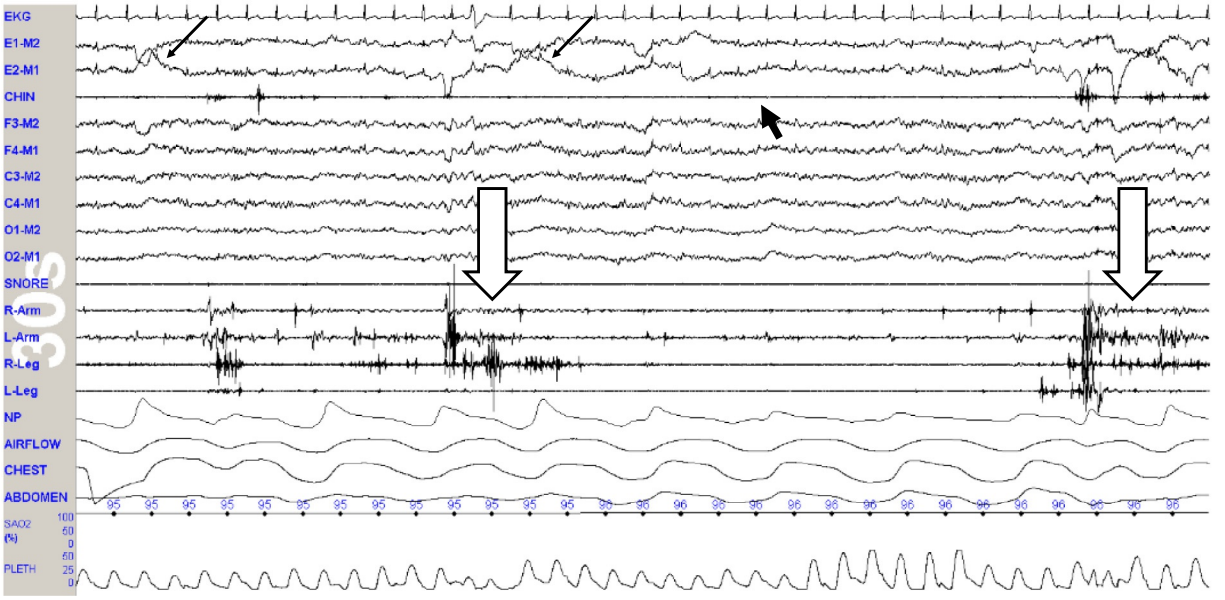

Fig. 25.7. Digital tracing of a 30-s epoch of stage REM sleep showing characteristic rapid eye movements (*thin arrows*) with low-chin tone (*thick arrow*). There is also elevated tone in the limb leads (*open arrows*), representing REM without atonia. This is a common finding in patients with REM sleep behavior disorder. E1-M2, left EOG; E2-M-1, right EOG; F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1, EEG derivations; chin, chin EMG; EKG, electrocardiogram; snore, snore microphone; R-Leg, right tibialis anterior EMG; L-Leg, left tibialis anterior EMG; R-arm, right extensor digitorum EMG; L-arm, left extensor digitorum EMG; NP, nasal pressure transducer; airflow, oronasal thermistor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage; pleth, plethysmography.

Table 25.1

Exclusion criteria for HSAT

1. Significant cardiorespiratory disease (i.e., congestive heart failure or chronic obstructive pulmonary disease)
2. Potential respiratory muscle weakness due to neuromuscular disease
3. Chronic opioid use
4. Awake or high risk for sleep-related hypoventilation (i.e., obesity hypoventilation)
5. Recent stroke
6. Severe insomnia
7. Symptoms of other sleep disorders (i.e., narcolepsy, central sleep apnea, movement disorders in sleep, parasomnias)
8. Environmental or personal reasons that may lead to poor acquisition and interpretation of HSAT results

of obesity hypoventilation syndrome, and without CO_2 monitoring, this diagnosis will be missed on HSAT.

PAP titration studies cannot be done using HSAT.

The value of HSAT over PSG in the diagnosis of OSA includes patient preference (Rosen et al., 2012), reduced cost, improved access to care, and insurance payor preference.

In the appropriate clinical settings, HSAT has been shown to be not inferior to PSG (Rosen et al., 2012; Berry and Sriram, 2014; Chai-Coetzer et al., 2014). Auto-adjusting PAP treatment for sleep apnea diagnosed by HST (Berry and Sriram, 2014) has been demonstrated. However, economic models assessing cost of diagnosis and treatment of OSA have not shown appreciable cost savings using HSAT.

Technology

HSAT devices typically use the same type of sensors used in PSG; however Type III (Level III) and Type IV (Level IV) sleep studies have fewer sensors in total, and EEG is not recorded. The most common devices used for HSAT are Type III devices, but others have extended sensors beyond the traditional Type III classification, using an alternative SCOPER (sleep, cardiovascular, oximetry, position, effort, and respiratory) assessment system (Collop et al., 2011). HSATs may also include sensors for body position, heart or pulse rate, movement assessment as a surrogate measure for sleep EEG for sleep, and pulse wave analysis, including peripheral arterial tonometry (Bar et al., 2003).

Type II devices have all of the same leads as Type I (in-lab PSGs) devices; however, no technician is present during the study to assist with technical concerns.

Type II devices are generally reserved for hospitalized patients. Type IV (Level IV) devices have one to two sensors, similar to nocturnal pulse oximetry testing. Type IV devices are not recommended by the AASM for HSAT, as these devices do not record oxygen saturation, airflow, or respiratory effort. Table 25.2 compares the sensors used in various types of sleep devices.

Particular brands of devices have not been specifically examined in research studies. Expense, patient comfort, ease of use, and integration with existing sleep-analysis systems are factors that influence the choice of technology. HSAT devices have been extensively reviewed and standards for the scoring of HSATs have been established (Collop et al., 2011).

Type III HSATs can detect obstructive respiratory events using the same types of airflow and effort sensors along with oxygen saturation information (Fig. 25.8).

HSAT program quality

Quality HSAT programs need to employ well-trained technologists who are skilled in the application of sensors required for accurate testing, and who can answer patient concerns, review data integrity, remove artifact, and accurately and reliably score records. Programs should have specific metrics measured as part of a quality assurance program. HSATs need to be interpreted by a board-certified sleep specialist. Programs that perform HSAT should have an established relationship with a sleep disorders center that provides comprehensive evaluation and testing services so that patients can have access to further evaluation and treatment as necessary.

Limitations of HSAT

The diagnosis of OSA may be missed or the severity may be underestimated using HSAT, as these devices do not measure sleep or EEG arousals during sleep. Therefore the total recording time is assumed to be equal to the total sleep time, which is seldom the case. As the total time spent asleep is part of the calculation of the AHI, this index would be lower using HSAT. A respiratory event index (REI) is preferred to the use of the AHI for HSAT. The REI is defined as the number of respiratory events per hour over the total recording time. Because EEG is not recorded, HSAT cannot measure REM sleep. Some patients have REM-predominant OSA with events occurring during REM sleep. These limitations may contribute to false negative results, or may severely underestimate the true AHI in REM sleep.

Research that has validated the use of HSAT has been performed in sleep centers, using research protocols, and not in the general population, in whom a lower pretest probability for OSA would be expected.

Additional limitations of HSAT include the tendency for the study to underdiagnose individuals with mild OSA, when comparing the measured AHI with PSG. HSAT in the home environment is associated with less supine sleep compared with the sleep lab setting, increasing the likelihood of a false negative test (Smith et al., 2007). Self-placement of sensors can be cumbersome and confusing, leading to poor quality recordings. Finally, the home environment may be less conducive to sleep than the lab if there are environmental disturbances to sleep, such as children or pets.

The failure rate of HSAT (Type III devices) from inadequate data is higher than with an attended in-lab PSG. One study demonstrated that with direct technologist involvement in the application of sensors the failure rate was as low as 7%, compared with 33% without (Golpe et al., 2003). Reviews have found that data loss sufficient to preclude an accurate HSAT interpretation using Level III devices was between 13% and 20% (Flemons et al., 2003). Research studies looking at failure rates typically have protocols with close follow-up that may not be reflective of the failure rate encountered in clinical practice. Additional limitations for generalizability of study results include variations in study design, entry criteria, comorbidities, age, body mass index, device used, and the number of subjects.

Failed tests necessitate repeat in-lab testing, limiting convenience to the patient and reducing cost savings. Patients who failed an initial HSAT had a higher failure on repeat HSATs (Rosen et al., 2012). Those patients had poorer follow through with the entire diagnostic process, resulting in 30% of subjects with technically inadequate HSATs and 16% of subjects with low AHI on HSAT who failed to proceed per protocol to PSG.

SUMMARY

PSG is considered to be the gold standard for the diagnosis of SRBD. Additionally, it can be used to evaluate a number of other sleep disorders, as well as to determine effectiveness of SRBD treatments. There are standardized methods and techniques for staging and scoring PSGs, which are included in the AASM scoring manual. Accredited sleep labs are required to follow AASM guidelines. HSAT can be utilized to confirm a diagnosis of OSA in patients who are considered to be at high risk for moderate to severe OSA without significant comorbid conditions; however, the possibility of a false negative test with HSAT is high.

Table 25.2

Comparison of sensors included in the various sleep study types

Sleep study types	Airflow					Respiratory effort		Optional leads										
	EEG	EOG	NT	Thermistor	PAP	Chest	Abdomen	Leg EMG	Chin EMG	ECG	HR	O_2 Sat	Body position	Snore	$EtCO_2$	$TcpCO_2$	Full EEG	Arm EMG
Type I devices																		
PSG	x	x	x	x		x	x	x	x	x	x	x	x	x	x	x	x	x
PAP titration	x	x			x	x	x	x	x	x	x	x	x	x		x	x	x
Split study	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
Type II device	x	x	x	x		x	x	x	x	x		x	x	x				
Type III device			x	x		x	x				x	x	x	x				
Type IV device											**x**	**x**						

EEG, electroencephalogram; *EOG*, electro-oculogram; *NT*, nasal pressure transducer; *PAP*, positive airway pressure; *EMG*, electromyogram; *ECG*, electrocardiogram; *HR*, heart rate; *O_2 sat*, oxygen saturation; *snore*, snore microphone; *$EtCO_2$*, end tidal carbon dioxide; *$TcpCO_2$*, transcutaneous carbon dioxide; *PM*, portable monitoring.

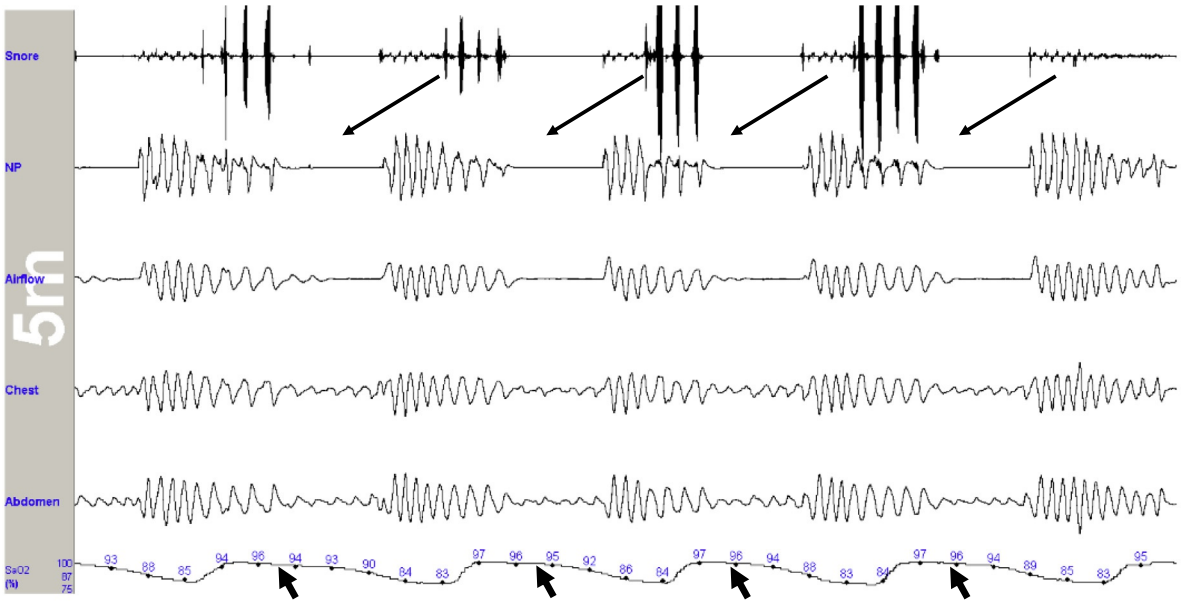

Fig. 25.8. Digital tracing of a 5-min epoch from a home sleep apnea test (HSAT) showing several obstructive apneas (*thin arrows*) with no flow in NP or airflow leads. Effort is present in chest and abdomen leads. There are also associated oxygen desaturations (*wide arrows*). NP, nasal pressure transducer; airflow, oronasal thermistor; chest, chest inductance plethysmography; abdomen, abdomen inductance plethysmography; SAO_2, oxygen saturation percentage.

References

Aldrich MS, Jahnke B (1991). Diagnostic value of video-EEG polysomnography. Neurology 41: 1060–1066.

Aserinsky E, Kleitman N (1953). Regularly occurring periods of eye motility and concomitant phenomena during sleep. Science 118: 273–274.

Bar A, Pillar G, Sheffy J et al. (2003). Evaluation of a portable device based on peripheral arterial tone for unattended home sleep studies. Chest 123 (3): 695–703.

Berger H (1929). Uber das elektroenkelphalogramm des Menshcen [About electroencephalogram of Menshcen]. Arch Psychiatr Nervenkr 87: 527–570 [in German].

Berry RB, Sriram P (2014). Auto-adjusting positive airway pressure treatment for sleep apnea diagnosed by home sleep testing. J Clin Sleep Med 10 (2): 1269–1275.

Berry RB, Brooks R, Gamaldo CE et al. (2017). For the American Academy of Sleep Medicine. In: The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Version 2.4, American Academy of Sleep Medicine, Darien, IL.

Boing S, Randerath WJ (2015). Chronic hypoventilation syndromes and sleep-related hypoventilation. J Thorac Dis 7 (8): 1273–1285.

Bramwell B (1909). Excessive sleepiness. Clini Stud Edinb 8: 276–277.

Broadbent WH (1877). Cheyne-stokes respiration in cerebral haemorrhage. Lancet 1: 307–309.

Carskadon MA, Dement WC (2017). Normal human sleep: an overview. In: M Kryger (Ed.), Principles and practice of sleep medicine, Elsevier, pp.15–24.

Caton R (1875). The electric current of the brain. Br Med J 2: 278.

Caton R (1889). Case of narcolepsy. Clin Soc Trans 22: 133–137.

Chai-Coetzer CL, Antic NA, Hamilton GS et al. (2014). Physician decision making and clinical outcomes with laboratory polysomnography or limited-channel sleep studies for obstructive sleep apnea: a randomized trial. Ann Intern Med 166 (5): 332–340.

Collop NA, Anderson WM, Boehlecke B et al. (2007). Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. J Clin Sleep Med 3: 737–747.

Collop NA, Tracy SL, Kapur V et al. (2011). Obstructive sleep apnea devices for out-of-center (OOC) testing: technology evaluation. J Clin Sleep Med 7 (5): 531–548.

Dement W, Kleitman N (1957). Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. Electroencephalogr Clin Neurophysiol 9 (4): 673–690.

Dement WC, Vaughan C (1999). The promise of sleep, Delacorte Press, New York, NY.

Dickens C (1837). The posthumous papers of the pickwickian club, Chapman and Hall, London.

Flemons WW, Littner MR, Rowley JA et al. (2003). Home diagnosis of sleep apnea: a systematic review of the literature. An evidence review cosponsored by the American Academy of Sleep Medicine, The American College of Chest Physicians, and the American Thoracic Society. Chest 124 (4): 1543–1570.

Foldvary-Schaefer N, Malow B (2011). Video recordings and video polysomnogram. In: P Montagna, S Chokroverty (Eds.), Handbook of clinical neurology. Sleep disorders, Part 1, 98. third series.

Fujita S, Conway W, Zorick F et al. (1981). Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg 89: 923–934.

Gastaut H, Tassinari CA, Duron B (1965). Polygraphic study of diurnal and nocturnal (hypnic and respiratory) episodal manifestations of Pickwick syndrome. Rev Neurol (Paris) 112 (6): 568–579 [in French].

Gastaut H, Tassinari CA, Duron B (1966). Polygraphic study of the episodic diurnal and nocturnal (hypnic and respiratory) manifestations of the Pickwick syndrome. Brain Res 2: 167–186.

Golpe R, Jimenez A, Carpizo R (2003). Home sleep studies in the assessment of sleep apnea/hypopnea syndrome. Chest 122 (4): 1156–1161.

Heitman SJ, Atkar RS, Hajduk EA et al. (2002). Validation of nasal pressure for the identification of apneas/hypopneas during sleep. Am J Respir Crit Care Med 166 (3): 386–391.

Iber C, Ancoli-Israel S, Chesson AL et al. (2007). The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications, American Academy of Sleep Medicine, Westchester, IL.

Jasper H (1958). The 10–20 electrode system of the International Federation. Electroencephalogr Clin Neurophysiol 10: 370–375.

Jouvet M, Michel F, Courjon J (1959). Sur un stade d'activite electrique cerebrale rapide au cours du sommeil physiologique [On a stage of rapid cerebral electrical activity in the course of physiological sleep]. CR Seances Soc Biol Fil 153: 1024–1028 [in French].

Jung R, Kuhlo W (1965). Neurophysiological studies of abnormal night sleep and the Pickwickian syndrome. Prog Brain Res 18: 140–159.

Kales A, Rechtschaffen A (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Association for the psychophysiological study of sleep, United States Government Printing Office, NIH Publication, Washington, DC.

Kapur VK, Auckley DH, Chowdhuri S et al. (2017). Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American academy of sleep medicine clinical practice guideline. J Clin Sleep Med 13 (3): 479–504.

Kuhlo W, Doll E, Frank C (1969). Erfolgreiche Behandlungeines Pickwick-Syndroms durch eine Dauertracheakanule. [Successful management of Pickwickian Syndrome using tracheostomy]. Dtsch Med Wochnschr 34: 1286–1290 [in German].

Kushida CA, Littner MR, Morgenthaler T et al. (2005). Practice parameters for the indications for polysomnography and related procedures: an update for 2005. Sleep 28: 499–521.

Lavie P (1984). Nothing new under the moon: historical accounts of sleep apnea syndrome. Arch Intern Med 144: 2025–2028.

Loomis AL, Harvey N, Hobart GA (1937). Cerebral states during sleep, as studied by human brain potentials. J Exp Psychol 21: 127–144.

McEvoy RD, Sharp DJ, Thornton AT (1986). The effects of posture on obstructive sleep apnea. Am Rev Respir Dis 133 (4): 662–666.

Minkel JD, Krystal AD, Benca RM (2017). Unipolar major depression. In: M Kryger (Ed.), Principles and practice of sleep medicine. Elsevier, 1352–1362.

Mitchell SW (1890). Some disorders of sleep. Am J Med Sci 100: 110–127.

Oksenberg A, Khamaysi I, Silverberg DS et al. (2000). Association of body position with severity of apneic events in patients with severe nonpositional obstructive sleep apnea. Chest 118 (4): 1018–1024.

Ong JC, Arnedt T, Gehrman PR (2017). Insomnia diagnosis, assessment and evaluation. In: M Kryger (Ed.), Principles and practice of sleep medicine. Elsevier, pp. 785–793.

Osler W (1918). Principles and practice of medicine, Butterworth, London.

Pelayo R, Hodgson N, Guilleminault C (2010). The history of sleep medicine. In: MJ Aminoff, F Boller, DF Swaab (Eds.), Handbook of clinical neurology. 95. Elsevier BV, Edimburgh-London, UK, pp. 547–556.

Redline S, Budhiraja R, Kapur V et al. (2007). The scoring of respiratory events in sleep: reliability and validity. J Clin Sleep Med 3 (2): 169–200.

Riley W, Powell NB, Guilleminault C et al. (1986). Maxillary, mandibular, and hyoid advancement: an alternative to tracheostomy in obstructive sleep apnea syndrome. Otolaryngol Head Neck Surg 94 (5): 584–588.

Rosen CL, Auckley D, Benca R et al. (2012). A multisite randomized trial of portable sleep studies and positive airway pressure autotitration versus laboratory-based polysomnography for the diagnosis and treatment of obstructive sleep apnea: the HomePAP study. Sleep 35 (6): 757–767.

Sateia M (Ed.), (2014). American Academy of Sleep Medicine. In: International classification of sleep disorders, third edn. American Academy of Sleep Medicine, Westchester, pp. 108–133. ICSD-3.

Silber MH (2009). Staging sleep. Sleep Med Clin 4 (3): 343–352.

Silber MH, Ancoli-Israel S, Bonnet MH et al. (2007). The visual scoring of sleep in adults. J Clin Sleep Med 3 (2): 121–131.

Silber MH, St Louis EK, Boeve BF (2017). Rapid eye movement parasomnias. In: M Kryger (Ed.), Principles and practice of sleep medicine, Elsevier, pp. 993–1001.

Smith LA, Chong DW, Vennelle M et al. (2007). Diagnosis of sleep-disordered breathing in patients with chronic heart failure: evaluation of a portable limited sleep study system. J Sleep Res 16: 428–435.

Sullivan CE, Issa FG, Berthon-Jones M et al. (1981). Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1: 862–865.

Thorpy MJ (2011). History of sleep medicine. In: MJ Aminoff, F Boller, DF Swaab (Eds.), Handbook of clinical neurology. 98. Elsevier BV, Amsterdam (Netherlands), pp. 3–25.

Williams RL, Karacan I, Hursch CJ (1974). EEG of human sleep: clinical applications, Wiley, New York.